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Effects of creatine supplementation on body composition, strength, and sprint performance

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Abstract

Purpose: To determine the effects of 28 d of creatine supplementation during training on body composition, strength, sprint performance, and hematological profiles.

Methods: In a double-blind and randomized manner, 25 NCAA division IA football players were matched-paired and assigned to supplement their diet for 28 d during resistance/agility training (8 h·wk^{-1}) with a Phosphagen HP (Experimental and Applied Sciences, Golden, CO) placebo (P) containing 99 g·d^{-1} of glucose, 3 g·d^{-1} of taurine, 1.1 g·d^{-1} of disodium phosphate, and 1.2 g·d^{-1} of potassium phosphate (P) or Phosphagen HP containing the P with 15.75 g·d^{-1} of HPCE pure creatine monohydrate (HP). Before and after supplementation, fasting blood samples were obtained; total body weight, total body water, and body composition were determined; subjects performed a maximal repetition test on the isotonic bench press, squat, and power clean; and subjects performed a cycle ergometer sprint test (12 × 6-s sprints with 30-s rest recovery).

Results: Hematological parameters remained within normal clinical limits for active individuals with no side effects reported. Total body weight significantly increased ($P < 0.05$) in the HP group (P 0.85 ± 2.2; HP 2.42 ± 1.4 kg) while no differences were observed in the percentage of total body water. DEXA scanned body mass (P 0.77 ± 1.8; HP 2.22± 1.5 kg) and fat/bone-free mass (P 1.33 ± 1.1; HP 2.43 ± 1.4 kg) were significantly increased in the HP group. Gains in bench press lifting volume (P -5 ± 134; HP 225 ± 246 kg), the sum of bench press, squat, and power clean lifting volume (P 1,105 ± 429; HP 1,558± 645 kg), and total work performed during the first five 6-s sprints was significantly greater in the HP group.

Conclusion: The addition of creatine to the glucose/taurine/electrolyte supplement promoted greater gains in fat/bone-free mass, isotonic lifting volume, and sprint performance during intense resistance/agility training.

During explosive sprinting exercise, the energy supplied to rephosphorylate adenosine diphosphate (ADP) to adenosine triphosphate (ATP) is determined largely by the amount of phosphocreatine (PC) stored in the muscle[10,31]. As PC stores become depleted, performance is likely to rapidly deteriorate because of the inability to resynthesize ATP at the rate required [29]. Since the availability of PC stores in the muscle may significantly influence the amount of energy generated during brief periods of high intensity exercise, it has been hypothesized that increasing muscle creatine content may increase the availability of PC and allow for an accelerated rate of resynthesis of ATP during and following high intensity, short duration exercises[4,22,24,28,32].

Studies that have evaluated these hypotheses have found that supplementing the diet with approximately 20 $g \cdot d^{-1}$ of creatine monohydrate for 2-7 d may elevate total creatine content in muscle by 10-20%, with 20-40% of the increased intramuscular creatine in the form of PC[4,5,20,22,24,28,32,38,41,47,51,52]. In addition, studies suggest that creatine supplementation may affect myocardial [11,19] and skeletal muscle metabolism by accelerating the rate of ATP resynthesis during and/or following repeated bouts of high-intensity exercise[5,22,24,38]. Theoretically this would improve repetitive sprint performance capacity.

In support of these hypotheses, studies indicate that creatine loading may improve high-intensity exercise performance in rowing[43], running [29,45], cycling[2,5,8,13,14,34], swimming [25], and resistance exercise[14,23,38,45,49]. Studies have also indicated that creatine supplementation may increase total body weight[2-5,14,22,38,40,49] and/or lean body mass [14,45], possibly because of fluid retention [4] and/or stimulating protein synthesis [7,33,44]. Finally, recent reports suggest that ingestion of carbohydrate with creatine enhances intramuscular creatine uptake [20,21] and glycogen deposition [21] and that creatine transport may be sodium dependent [26]. Consequently, ingestion of glucose and sodium with creatine would theoretically provide additional ergogenic benefit.

However, not all studies investigating the ergogenic value of creatine supplementation have reported enhanced exercise performance[(3,6,9,12,16,40-42,46)]. In addition, while increases in total body weight have been reported in most studies, the effects of creatine supplementation on body composition are less clear. Finally, although no side effects have been reported other than weight gain [(3)], little data are available evaluating the medical safety of supplementing the diet with creatine during training for prolonged periods of time. The purpose of this study was 1) to examine the effects of ingesting a supplement designed to enhance creatine uptake during intense resistance/agility training on body composition, maximal lifting volume, and sprint performance in well-trained athletes; and 2) to evaluate the effects of 28 d of creatine supplementation on clinical chemistry profiles.

Back to Top | Article Outline

MATERIALS AND METHODS

Subjects. Twenty eight NCAA division IA football players undergoing winter/spring off-season resistance/football training at a major university in the mid-south region of the United States volunteered to participated in this study. Subjects were informed as to the experimental procedures and signed informed consent statements in adherence with the human subjects guidelines of The University of Memphis and the American College of Sports Medicine. Twenty five subjects who were descriptively (mean ± SEM) 19.9 ± 0.3 yr, 97.2 ± 4 kg, and 183 ± 2 cm completed the study. Remaining subjects were unable to complete the study because of injury, illness, and/or inability to comply with the study protocol.

Subjects signed statements indicating that they were not taking anabolic steroids and that they were aware that they may be subject to random drug testing during the study according to NCAA regulations. During the conduct of the study, 16 subjects were randomly selected by the NCAA for drug testing during two independent screenings. All drug tests were negative for the presence of anabolic/androgenic steroids according to NCAA criteria.

Experimental design. Subjects maintained their normal diet throughout the study. Meals consisted of *ad libitum* intake of a primary entree and a limited number of side entrees served at the team training table meals. Consequently, although the athletes were allowed to select their own foods and ingest food outside of the training table, diets of the athletes were similar. Moreover, subjects were not allowed to have ingested creatine, β-hydroxy β-methylbutyrate (HMB), or beta-agonists for an 8-wk period before the start of supplementation. Subjects were also instructed not to ingest any other nutritional supplements, proposed ergogenic aids, or nonprescription drugs during the course of the study.

During the first 2 wk of training, subjects participated in two familiarization sessions and completed presupplementation assessments. In the first familiarization session, the procedures of the study were explained, the subjects were weighed, and training and medical history forms were completed. In addition, the subjects practiced the cycle ergometer sprint test to be used in the study. This involved being seated on the CardiO_2 computerized cycle ergometer (ErgometRx, St. Paul, MN) and having the subjects perform 12× 6-s maximal effort sprints with 30 s of rest between each sprint at a standardized work rate. Subjects performed one additional practice sprint trial before presupplementation testing. In addition, subjects were instructed how to report nutritional intake on nutritional log sheets by a co-investigator trained in clinical nutrition.

Presupplementation assessments included: 1) a 4-d nutritional intake assessment (including one weekend day); 2) donation of an 8-h fasting venous blood sample; 3) measuring total body weight, body water, and body composition; 4) performance of low repetition maximal effort isotonic bench press and squat tests; and 5) performance of a 12 × 6-s sprint test on the cycle ergometer with 30-s rest recovery between sprints.

In a double-blind and randomized manner, subjects were then matched by total body weight and assigned to supplement their diet for 28 d with either a Phosphagen HP placebo (P) containing 99 $g \cdot d^{-1}$ of glucose, 3 $g \cdot d^{-1}$ of taurine, 1.1 $g \cdot d^{-1}$ of disodium phosphate, and 1.2 $g \cdot d^{-1}$ of potassium phosphate ($N = 14$) or Phosphagen HP (Experimental and Applied Sciences, Inc, Golden, CO) containing the P formulation with 15.75 $g \cdot d^{-1}$ of HPCE pure creatine monohydrate (HP; $N = 11$). Supplements were prepared in powder form with similar texture, taste, and appearance and were independently packaged in generic foil packets for double-blind administration. Subjects mixed the supplement powder into approximately 0.25 L of water and ingested the solution with morning, mid-day, and evening meals.

Supplement packets were administered in blindly coded boxes containing a 15-d supply of the supplements. Subject compliance in taking the supplements was verified by having a research assistant collect empty supplement packets at evening meals throughout the study. Subjects had to turn in all empty packets to receive the next 15-d supply of supplements. In addition, subjects had to turn in all empty packets throughout the remainder of the study to receive the incentive for participating in the study (i.e., four cans of Phosphagain, Experimental & Applied Sciences). Consequently, compliance in taking the supplements was excellent.

During the 4-wk supplementation period, subjects participated in a standardized resistance and agility training program. The program consisted of 5 $h \cdot wk^{-1}$ of heavy resistance training conducted on Monday, Tuesday, Thursday, and Friday afternoons, as well as a 3 $h \cdot wk^{-1}$ of agility/sprint training conducted at 6:00 a.m. on Monday, Wednesday, and Friday mornings. Primary lifts performed included bench press, incline bench press, shoulder press, lateral pull downs, seated cable rows, upright rows, abdominals, squats, hip sled, gluteal/hamstring raises, power hang cleans, and clean and jerk. Lifts were prescribed in a structured program on a weekly rotation of lifts/sets/repetitions within a 4-wk microcyle (e.g., 1 to 3 sets of two to eight repetitions at intensities ranging from 60 to 95% of 1 RM(repetition maximum)). Agility training consisted of high intensity sprint and football agility drills. All training was performed under the supervision of certified strength coaches and/or assistant football coaches. Attendance was monitored and subjects who missed workouts were required to make them up according to team policy.

Following the 28-d supplementation period, subjects underwent postsupplementation assessments in a similar manner as the presupplementation tests. Therefore, diet was recorded for 4 d; subjects donated a fasting venous blood sample; body weight, body water, and body composition were determined; subjects performed the maximal effort low repetition test on the isotonic bench press and squat; and the subjects performed the 12 × 6-s cycle ergometer sprint test with 30-s recovery between sprints.

Nutritional intake was monitored for 4 d before the initiation of supplementation and during the final week of supplementation. This was accomplished by having a registered dietitian and research assistants evaluate and record all food/fluid ingested during training table meals. In addition, subjects reported any additional food/fluids ingested between meals during this period. Nutritional records were analyzed by a registered dietitian using the Food Processor III nutritional analysis software (Nutritional Systems, Salem, OR).

Subjects observed an overnight 8-h fast before donating blood samples. Venous blood samples were obtained between 6:00 and 7:30 a.m. via venipuncture from an antecubital vein in the forearm using standard phlebotomy procedures. Venous blood was collected into 10-mL serum separation tubes (SST) and a 5-mL tube containing K_3. The SST tubes were centrifuged at 5,000 rev·min^{-1} for 10-min using a Biofuge 17R centrifuge (Heraeus Inc., Germany). Samples were refrigerated and then shipped overnight in cold containers to Corning Clinical Laboratories (St. Louis, MO) for clinical analyses. A complete clinical chemistry panel (31 items) was run on serum samples using the Technicon DAX model 96-0147 automated chemistry analyzer using standard clinical procedures (Technicon Inc., Tarrytown, NY). Cell blood counts with percent differentials were run on whole blood samples using a Coulter STKS automated analyzer using standard procedures (Coulter Inc., Hialeah, FL).

Total body weight was measured on a calibrated digital scale with a precision of ± 0.02 kg (Sterling Scale Co., Southfield, MI). Total body water was estimated [(48)] using a Valhalla 1990b Bioelectrical Impedance Analyzer (San Diego, CA).

Whole body (excluding cranium) body composition measurements were determined using a Hologic QDR-2000 dual energy x-ray absorptiometer (Waltham, MA) with the Hologic version V 7, REV *F* software. This system measures the amount of bone, fat, and fat-free/soft tissue mass which falls within standardized density ranges using dual energy x-ray absorptiometry methodology (DEXA). The DEXA scans regions of the body (right arm, left arm, trunk, right leg, and left leg) to determine the amount of bone mass, fat mass, and fat-free/soft tissue mass within each region. The scanned bone, fat, and fat-free/soft tissue mass for each region are then subtotaled to determine whole body (excluding cranium) values. Percent body fat is calculated by dividing the amount of measured fat mass by total scanned mass (sum of bone mass, fat mass, and fat-free/soft tissue mass). It should be noted that the DEXA does not consider total body weight when the densities of bone, fat, and fat-free/soft tissue mass are determined. The DEXA records only the amount of tissue measured within the standardized density ranges. Therefore, the scanned total body mass (adding an estimate for the cranium mass) may not equal total body weight. DEXA has been shown to be a highly reliable (r = 0.99) and precise method (coefficient of variation of 0.5-1%) for determining individual body composition segments[(18,30,35,39)].

Quality control (QC) calibration procedures were performed on a spine phantom (Hologic X-CALIBER Model DPA/QDR-1 anthropometric spine phantom) before each testing session. The spine phantom was scanned in the AP single-beam mode, array mode, and lateral array modes. Analysis of QC scans were performed by comparing daily QC scans with a Hologic reference scan of the phantom. Data from the daily QC scans were then entered into the database and compared with factory values. Values for daily QC scans were plotted for both bone mineral content (BMC) and bone mineral density (BMD) in all mentioned modes. Tolerance levels for the QC scans were set at ± 1 SD from the unit mean, which is determined by Hologic for each individual unit. Mean coefficients of variation in BMC and BMD measurements obtained in the lateral and array modes ranged between 0.41 to 0.55% throughout the life of the unit. Subjects were positioned according to standardized criteria during the initial scan. This position was referenced into the computer for positioning of subjects in subsequent trials. DEXAs were performed by technicians certified in radiology to perform DEXAs.

Subjects performed maximal effort repetition tests on the isotonic bench press, squat, and power clean to determine lifting volume. This strength testing approach was selected in consultation with the strength coaches because it more closely represented the type of resistance training the athletes were involved in during the study (i.e., a 4-wk periodized cycle of mid-range repetitions). Basically, this involved having the athletes warm up and then perform a maximal effort repetition test with a weight that the strength coaches estimated the athlete could lift between four and eight times based on training lifting performance. Lifting volume was determined by multiplying the amount of weight lifted by the number of repetitions performed. Total lifting volume was determined by adding the sum of bench press, squat, and power clean lifting volumes. All isotonic test sets were performed under supervision of certified strength coaches using standardized lifting criteria [(17,36,50)].

The sprint tests were performed on a computerized CardiO_2 cycle ergometer equipped with toe clips at a standardized work rate of 3.85 $J \cdot kg^{-1} \cdot rev^{-1}$ (ErgometR_x Corp., St. Paul, MN). Seat position was standardized between trials. The ergometer was connected via an RS232 parallel interface to a Dell 466/Le Optiplex computer(Dell Computer Corp., Austin, TX) using ErgometR_x Cardioscribe and Exerscribe software (ErgometR_x Corp.). Crank frequency was measured using a crystal referenced optic encoder with a precision range of 0-200 $rev \cdot min^{-1}$ and an accuracy of ± 1 $rev \cdot min^{-1}$. Pedal torque was determined by a calibrated strain gauge with a range of 0 to 2,000 W and an accuracy of ± 1%. Data were collected and downloaded into the computer at 0.5-s intervals.

Statistical analysis. Nutritional, hematological, body composition, and strength data were analyzed by a 2 × 2 repeated measures ANOVA using SPSS for Windows Version 7.5 software. Delta scores (post and pre values) were calculated on selected variables and analyzed by one-way ANOVA. To normalize differences between groups in presupplementation sprint performance, Day 28 work data were analyzed by ANCOVA using Day 0 data as the covariate. Tukey *post hoc* procedures were performed on the adjusted means to examine significant group × sprint effects. Data are presented as means ± SDs of means. Data were considered significantly different when the probability of error was 0.05 or less.

Back to Top | Article Outline

RESULTS

Side effects. Subjects tolerated the supplementation protocol well with no reports of gastrointestinal distress and/or medical problems/symptoms. In addition, there was no evidence of muscular cramping during resistance/agility training sessions or during performance trials.

Nutritional intake. No significant differences were observed between groups in mean estimated total energy intake (P 42 ± 10; HP 39± 8 $kcal \cdot kg^{-1} \cdot d^{-1}$, $P = 0.49$), carbohydrate intake (P 5.3 ± 0.9; HP 4.8 ± 1.0 $g \cdot kg^{-1} \cdot d^{-1}$, $P = 0.14$), fat intake (P 1.5± 0.4; HP 1.6 ± 0.4 $g \cdot kg^{-1} \cdot d^{-1}$,$P = 0.62$), or protein intake (P 1.7 ± 0.6; HP 1.6 ± 0.4 $g \cdot kg^{-1} \cdot d^{-1}$, $P = 0.70$).

Clinical chemistry profiles. All blood variables evaluated remained within normal limits for individuals engaged in heavy exercise training. No significant group × time interactions were observed in plasma glucose, carbon dioxide, urea nitrogen, uric acid, total protein, albumin, alkaline phosphatase, sodium, potassium, chloride, calcium, ionized calcium, phosphorus, leukocytes, neutrophils, lymphocytes, monocytes, eosonophils, basophils, hemoglobin, hematocrit, total bilirubin, total iron, platelets, red blood cells, red blood cell distribution width, mean corpuscular volume, or mean platelet volume.

Tables 1, 2, and 3 present remaining blood variables evaluated. Creatinine and globulin levels were significantly increased in the HP group, whereas the ratio of blood urea nitrogen/creatinine was significantly increased in the P group. In addition, the ratio of albumin/globulin was significantly decreased in the HP group. Results also provide some evidence of a mild elevation in creatine kinase (CK), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) activities in the HP group with no effects on g-glutamyltransferase (GGT). Subjects in the HP group also exhibited some evidence of an improved lipid profile (i.e., 13% increased in HDL, a 13% decrease in VLDL, and a 7% decrease in the ratio of HDL/CHOL).


Table 1
Image Tools


Table 2
Image Tools


Table 3
Image Tools

Total body weight and body water. Total body weight was significantly increased in both groups. However, gains in total body weight were significantly greater in the HP group (P 0.85 ± 2.2; HP 2.42± 1.4 kg, $P = 0.05$). No significant differences were observed between groups in total body water changes expressed as a percentage of total body weight (P -0.04 ± 0.7; HP -0.01 ± 0.6%, $P = 0.93$).

Body composition. Table 4 presents DEXA determined body composition data obtained on days 0 and 28 of supplementation, while Figure 1 presents mean changes in body composition values from Day 0. No significant differences were observed in mean changes in bone mass, fat mass, or percent body fat between the P and HP groups. Gains in scanned total body mass and fat/bone-free mass were significantly greater in the HP group.


Table 4
Image Tools

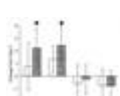

Figure 1-Changes in ...
Image Tools

Strength. Subjects in the HP group observed a significantly greater increase in bench press lifting volume (P -5 ± 134; HP 225± 246 kg, *P* = 0.002). No significant differences were observed in changes in squat (P 267 ± 308; HP 327 ± 350 kg,*P* = 0.56) or power clean lifting volume (P 921 ± 326; HP 1100± 485 kg, *P* = 0.28). However, changes in total lifting volume(sum of bench press, squat, and power clean) were significantly greater in the HP group (P 1,105 ± 429; HP 1,558 ± 645 kg, *P* = 0.05).

Sprint performance. Figure 2 presents Day 0 and Day 28 mean work (J) responses observed for the P and HP groups during the 12 × 6-s sprints, while Figure 3 presents mean changes in work (J) for the 12 × 6-s sprint test. ANCOVA revealed a significant group × sprint effect (*P* = 0.006) in Day 28 work values. *Post hoc* analysis revealed that work was significantly greater in the HP group during sprints 1 to 5, with differences between groups dissipating thereafter. Mean improvement in total accumulated work to perform the 12 × 6 sprints was 51% greater in the HP group; however, this difference was not significantly different between groups (P 4,599 ± 7,427; HP 6,951 ± 7,718 J, *P* = 0.45).


Figure 2-Pre- () and...
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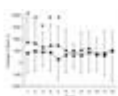
Figure 3-Mean change...
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Back to Top | Article Outline

DISCUSSION

Results of the present study indicate that 28 d of creatine supplementation(15.75 $g \cdot d^{-1}$) with glucose, taurine, and electrolyte during resistance/agility training promoted significantly greater gains in fat/bone-free mass, isotonic lifting volume, and sprint performance in comparison with ingestion of the glucose, taurine, and electrolyte formulation alone. These findings support previous reports that creatine supplementation may increase total body weight[(2-5,14,20,38,40,49)] and/or lean body mass [(14,45)], promote greater gains in strength [(14,45,49)], and improve intense exercise performance and/or recovery[(2,5,13,14,22,24,25,29,33,38,43,45)]. Although the etiology of these improvements remains to be determined, our results provide additional evidence that creatine supplementation may enhance physiological adaptations to resistance/agility training.

Clinical chemistry responses. Although creatine supplementation has become a popular nutritional supplement among athletes[(35)], little is known regarding the medical safety of short-term (<7-d) or long-term (>7-d) creatine supplementation during training at the proposed ergogenic doses (i.e., 10-20 $g \cdot d^{-1}$). Short-term creatine supplementation has been reported to increase erythrocytic creatine content and mildly increase serum creatine[(20,21,28,32)] and creatinine levels[(32)]. However, studies conducted in an older subject population over an 8-wk period, at daily doses ranging from 10-20 g, found no significant effects on creatinine concentrations. In the present study, ingestion of creatine with glucose, taurine, and electrolytes for 28 d resulted in a small but significant increase in fasting serum creatinine levels. Clinically, serum creatinine is used as an indirect marker of renal stress [27]. Normal fasting serum creatinine concentrations in untrained subjects typically range between 90-110μmol·L^{-1} but may increase up to ten-fold under certain pathological conditions [27]. Intense exercise typically increases serum creatinine levels by 20 to 50 μmol·L^{-1} as well as promotes urinary creatinine excretion[4,10,20,21,32]. The exercise-induced increase in serum and urinary creatinine levels has been suggested to reflect an increased release and cycling of intramuscular creatine as a consequence of myofibrillar protein turnover[4,10,15]. While the concentrations observed after HP supplementation remained within normal limits for individuals engaged in intense training (i.e., 100 to 150μmol·L^{-1}), results suggest that creatine supplementation(15.75 g·d^{-1} for 28 d) may increase fasting serum creatinine levels. The etiology and clinical significance of the mild elevation in serum creatinine levels remains to be determined.

Second, creatine supplementation was associated with a moderate increase in muscle and liver enzyme efflux. In active populations, elevations in creatinine and muscle/liver enzymes are typically used as indicators of training/exercise intensity [4,10,27]. In this regard, the more intense the training/exercise bout, the higher the muscle and liver enzyme efflux. Consequently, it could be argued that although the subjects underwent similar training, subjects in the HP group may have been able to maintain a greater training intensity during the program, promoting greater muscle/liver enzyme efflux. However, long-term supplementation (8-wk) of creatine in older individuals not participating in intense training also has been associated with moderate increases in CK concentrations [1]. Interestingly though, the ratio of urea nitrogen/creatinine was unchanged in the HP group while being significantly elevated in the P group. Increases in the ratio of urea nitrogen/creatinine are used as a general marker of catabolism[27]. Consequently, this finding suggests that despite the moderate increases in serum muscle and liver enzyme efflux observed, subjects in the HP group may have experienced less catabolism and/or greater nitrogen retention during training in comparison with the placebo group. This finding supports contentions that creatine supplementation may allow the athlete to maintain a greater training volume/intensity and thereby improve the quality of training. Additional research should evaluate the effects of creatine supplementation on training volume, training intensity, muscle/liver enzyme efflux, and markers of skeletal muscle proteolysis.

Third, analysis of blood lipid data provides some additional evidence that creatine supplementation may affect blood lipids. In this regard, HDL concentrations were significantly increased (13%) while there was some evidence that VLDL levels (-13%) and the ratio of total cholesterol to HDL levels (-7%) decreased in the creatine group. These findings support the report of Earnest et al. [15] that creatine supplementation significantly decreased total cholesterol, triglycerides, and VLDL in moderately hyperlipidemic, physically active male and female subjects. Additional research should evaluate the potential lipid modulating effects of creatine supplementation.

Body composition. Short-term creatine supplementation (5 to 7 d) has been reported to increase total body mass by approximately 0.6 to 1.1 kg[2-5,14,20,38,40,49]. The increase in total body mass has been suggested to be a result of an increase in total body water content [4] and/or a creatine-stimulated increase in myofibrillar protein synthesis[7,33,44]. While we are not aware of any studies reporting a disproportionate increase in total body water content following creatine supplementation, studies have reported that creatine promotes amino acid uptake and stimulates myofibrillar protein synthesis[7,33]. Moreover, there is evidence in patient populations that depletion of intramuscular creatine is associated with atrophy of Type II muscle fibers and that creatine supplementation (1.5 $g \cdot d^{-1}$ for 1 yr) in creatine depleted gyrate atrophy patients significantly increased total body weight by 10% and Type II muscle fiber diameter by 34% [44].

Unfortunately, few studies have evaluated the effects of creatine supplementation on body composition. Earnest et al. [14] reported that 28 d of creatine supplementation (20 $g \cdot d^{-1}$) during resistance training significantly increased total body mass by 1.7 kg($P < 0.05$) and that gains in hydrostatically determined fat-free mass accounted for 1.5 kg of the total body mass gain ($P = 0.054$). In addition, Stout et al. [45] found that 8 wk of creatine supplementation (21 $g \cdot d^{-1}$ for 5 d and 10.5 $g \cdot d^{-1}$ for 51 d) during off-season football resistance/agility training did not significantly increase DEXA determined fat-free mass (2.6± 2.0 kg) in comparison with a glucose placebo (-0.01 ± 2.6 kg). However, addition of glucose, taurine, and electrolytes to the creatine supplement (Phosphagen HP as used in the present study) promoted significant increases in fat-free mass (2.9 ± 1.5 kg) in comparison with those in the glucose placebo.

In the present study, creatine supplementation with glucose, taurine, and electrolytes promoted significantly greater gains in total body mass (P 0.85± 2.2; HP 2.42 ± 1.4 kg), scanned body mass (P 0.77 ± 1.8; HP 2.22 ± 1.5 kg), and fat/bone-free mass (P 1.33 ± 1.1; HP 2.43 ± 1.4 kg) in comparison with ingestion of the glucose, taurine, electrolyte supplement alone. Moreover, the increases in body mass could not be explained by disproportionate increases in total body water content as determined by bioelectrical impedance. These findings provide additional evidence that creatine supplementation may promote lean tissue accretion during resistance/agility training and that ingestion of creatine with glucose, taurine, and electrolytes may promote greater gains in fat/bone-free mass (2.43 ± 1.4 kg) than previously reported[14] with creatine supplementation alone (i.e., 1.5 kg). However, additional research is necessary to evaluate the effects of creatine supplementation on body composition, fluid retention/total body water content using more precise methods, and protein synthesis. Moreover, additional research should evaluate the potential additive and/or synergistic effects that creatine, glucose, taurine, sodium phosphate, and potassium phosphate may have on lean tissue accretion during training.

Performance. The majority of studies investigating the effects of creatine supplementation have focused on the potential ergogenic value during exercise following short-term loading periods (i.e., 15-20 $g \cdot d^{-1}$ for 5-7 d). These studies have indicated that creatine supplementation improved: 1000-m rowing performance [43]; repetitive running [29], cycling[2,5,8,13,14,34], and swimming [25] sprint performances; the amount of work performed during repeated sets of isokinetic contractions[23,38]; and gains in strength/lifting volume[38,49]. However, other studies have reported no ergogenic value on single-sprint performance in swimmers[9,40], 60-m sprint capacity in field hockey and baseball players [42], cycling sprint performance[6,12,16,41], submaximal endurance exercise [46], or high-intensity endurance running[3].

Fewer studies have investigated the effects of longer periods of creatine supplementation on exercise performance. Grindstaff et al.[25] found that 9 d of creatine supplementation (21 $g \cdot d^{-1}$) significantly improved swim performance times during 3× 100-m freestyle swims with 60-s rest recovery and arm ergometer performance when performing 3 × 20-s sprints with 60-s rest recovery in competitive junior swimmers. Earnest et al. [14] found that 28 d of creatine supplementation (20 $g \cdot d^{-1}$) during resistance training significantly increased: 3 × 30-s maximal effort cycling performance with 5-min rest recovery between bouts; 1 RM bench press performance; and bench press lifting volume at 70% of 1 RM.

Similarly, Stout et al. [(45)] reported that 8 wk of creatine supplementation (21 $g \cdot d^{-1}$ for 5 d and 10.5 $g \cdot d^{-1}$ for 51 d) did not significantly improve changes 1 RM bench press (placebo 13.1 ± 9.6; creatine 17.5 ± 9.3 kg), vertical jump performance (placebo 1.3 ± 3.5; creatine 5.1 ± 3.8 cm), or 100-yd sprint times (placebo -0.02 ± 0.09; creatine -0.24 ± 0.16-s) during off-season football resistance/agility training. However, addition of glucose, taurine, and electrolytes to the creatine supplement(Phosphagen HP) promoted significant increases in 1 RM bench press (28.8± 13.0 kg), vertical jump performance (5.6 ± 2.9 cm), and 100-yard sprint times (-0.31 ± 0.1 s) in comparison with those of the placebo.

Results of the present study support these findings in that subjects in the creatine supplemented group had greater gains in bench press lifting volume and cycle ergometer sprint performance during the first five 6-s sprints. Collectively, these findings suggest that 9-56 d of creatine supplementation may enhance the quality of training leading to improved repetitive sprint performance and/or strength. However, additional research is necessary to determine whether the improved sprint and strength performance would result in greater performance in various athletic events.

Back to Top | Article Outline

SUMMARY

Results indicate that 28 d of creatine supplementation (15.75 $g \cdot d^{-1}$) with glucose, taurine, and electrolytes promoted significantly greater gains in fat/bone-free mass, upper extremity lifting volume, and sprint performance during resistance/agility training in well-trained athletes in comparison with ingesting the glucose, taurine, and electrolyte formulation alone. These findings support previous reports that creatine supplementation may provide ergogenic benefit. Additional research should evaluate: 1) the role of creatine supplementation on protein turnover, serum creatinine kinetics, muscle and liver enzyme efflux, lipid and cholesterol metabolism, fluid retention/total body water, and lean tissue accretion; 2) the effects of creatine supplementation on training volume/intensity and performance in a variety of sports events; 3) the medical safety of long-term supplementation of creatine; and 4) the additive and/or synergistic role that creatine, glucose, taurine, sodium phosphate, and potassium phosphate may have on body composition and performance.

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interest in the outcome of results reported. A. L. Almada is cofounder and a consultant for Experimental and Applied Sciences, Inc. and served as a consultant and liaison between investigators at The University of Memphis and the granting agency. Presentation of results in this study does not constitute endorsement of the product investigated by The University of Memphis nor the American College of Sports Medicine.

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Back to Top | Article Outline

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Back to Top | Article Outline
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